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United States Department of State

Chemical Warfare in Southeast Asia and Afghanistan: An Update

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Report from
Secretary of State
George P. Shultz

November 1982

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THE SECRETARY OF STATE
WASHINGTON

TO THE CONGRESS OF THE UNITED STATES
AND MEMBER STATES OF THE UNITED NATIONS:

Chemical and toxin weapons are of special concern to mankind. Used against civilian populations, or even against soldiers with protective equipment, they can cause protracted and indiscriminate physical and psychological suffering and, as we witnessed in World War I, widespread death as well. For such reasons, the international community has outlawed the use of these weapons. The 1925 Geneva Protocol, one of the oldest arms control agreements still in force, forbids the use of chemical and biological weapons in war. The 1972 Biological and Toxin Weapons Convention prohibits the mere possession of toxin weapons. In an effort to extend such legal constraints still further, the United States—together with other countries in the Committee on Disarmament at Geneva—is seeking an outright ban on the development, production and stockpiling of chemical weapons.

I regret, then, to report that chemical and toxin weapons are nevertheless being used today in Laos, Kampuchea and Afghanistan by the Soviet Union and its allies. In March of this year, we reported on this subject to the Congress, the United Nations, and to the members of the international community. Our report, which contained a comprehensive and detailed compilation of the evidence available to the United States Government, was designed to bring the issue to the attention of the world community. In presenting it, we invited others to join us in examining the evidence and in confirming the truth.

These efforts have not led the Soviets and their allies to halt their illegal use of chemical and toxin weapons. Instead, they continue to deny the truth about their illegal activities. The world cannot be silent in the face of such human suffering and such cynical disregard for international law and agreements. The use of chemical and toxin weapons must be stopped. Respect for existing agreements must be restored and the agreements themselves strengthened. Respect for the dignity of humanity must be restored. Failure to achieve these goals can only have serious implications for the security of the world community, particularly for the security of smaller nations, like those whose people are being attacked. If such basic elements of human rights can be so fundamentally ignored, how can we believe any pledges to respect human rights?

All who would seek to promote human rights, and all who would seek to maintain the credibility of international agreements, have a duty to call world attention to the continuing use of chemical and toxin weapons, and to seek a halt to their use. It is for this purpose that the United States provides the following report.

Sincerely,

George P. Shultz

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This report presents conclusions based on further evidence about chemical and toxin warfare activities in Laos, Kampuchea, and Afghanistan that has become available to the U.S. Government since publication of the special report on this subject on March 22, 1982. The evidence includes new information on events occurring since the first of this year as well as additional information from a variety of sources on activities described in that report. The current report is accompanied by tables listing recent and newly reported attacks and annexes providing additional sample analysis results, medical evidence, and other supporting data.

UPDATED FINDINGS

Based on a thorough analysis of this new information, we are able to conclude the following:

- Reports of chemical attacks from February through October 1982 indicate that Soviet forces continue their selective use of chemicals and toxins against the resistance in Afghanistan. Moreover, new evidence collected in 1982 on Soviet and Afghan Government forces' use of chemical weapons from 1979 through 1981 reinforces the previous judgment that lethal chemical agents were used on the Afghan resistance. Physical samples from Afghanistan also provide new evidence of mycotoxin use.
- Vietnamese and Lao troops, under direct Soviet supervision, have continued to use lethal and incapacitating chemical agents and toxins against the H'Mong resistance in Laos through at least June 1982.
- Vietnamese forces have continued to use lethal and incapacitating chemical agents and toxins against resistance forces in Kampuchea through at least June 1982.
- Trichothecene toxins were found in the urine, blood, and tissue of victims of "yellow rain" attacks in Laos and Kampuchea and in samples of residue collected after attacks.
- We continue to find that a common factor in the evidence is Soviet involvement in the use of these weapons in all three countries. Continued analysis of prior data and newly acquired information about Soviet mycotoxin research and development, chemical warfare training in Vietnam, the presence of Soviet chemical warfare advisers in Laos and Vietnam, and the presence of the same unusual trichothecene toxins in samples collected from all three countries reinforce our earlier conclusion about the complicity of the Soviet Union and about its extent.

INTRODUCTION

Our March study showed that casualties and deaths resulted from chemical attacks in Southeast Asia and Afghanistan and that trichothecene toxins were used in both Laos and Kampuchea. The new evidence shows that these attacks are continuing in all three countries and that trichothecene toxins have been used in Afghanistan as well.

The same rigorous analytical processes employed in our March study, and outlined in detail there, were followed to arrive at the judgments contained in this update. In light of the widespread publicity given the March report, special efforts were made by U.S. Government analysts to preclude being led astray by any possible false reports that might be generated for propaganda or other purposes and to eliminate the possibility of making erroneous judgments about the chemical or toxin agents involved because of tampering or improper handling. Every report has been carefully checked.

The evidence in the March study was based on a broad range of data, including testimony by physicians, refugee workers, journalists, and others. Although some of the new reports are anecdotal, we have been able to corroborate most of them by other sources and sample analysis. Moreover, personal testimony tends to add credence to other accounts which, taken together, form a coherent picture. The material presented in this report represents only a relatively small amount of the total accumulated evidence. This additional information is examined in greater detail in the annexes. Improved sample collection procedures, a better quality of medical histories and physical examinations, documentation including photographs of lesions and hospital charts from Southeast Asia, and interviews by trained personnel have reinforced our earlier conclusions and led to new discoveries.

As international concern about this subject has increased, based on the

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development of evidence from many countries, independent analyses have been initiated by foreign chemical warfare experts, physicians, journalists, and independent nongovernmental scientists and laboratories. Analysts in the United States have found this research very helpful both in supporting their own conclusions and, more importantly, in expanding on them.

SUMMARY OF EVIDENCE

Afghanistan

Evidence indicates that the Soviets have continued the selective use of toxic agents in Afghanistan as late as October 1982. For the first time we have obtained convincing evidence of the use of mycotoxins by Soviet forces through analyses of two contaminated Soviet gas masks acquired from Afghanistan. Analysis and quantification of material taken from the outside surface of one mask have shown the presence of trichothecene mycotoxin. Analysis of a hose from a second Soviet mask showed the presence of several mycotoxins. In addition, a vegetation sample from Afghanistan shows preliminary evidence of the presence of mycotoxins. (See Annex A.)

Our suspicions that mycotoxins have been used in Afghanistan have now been confirmed. Reports during 1980 and 1981 described a yellow-brown mist being delivered in attacks which caused blistering, nausea, vomiting, and other symptoms similar to those described by "yellow rain" victims in Southeast Asia. Because of limited access to survivors who still exhibited symptoms, as well as great difficulties in collecting environmental and other physical samples from attack sites, we were unable to conclude with certainty in the March 22 report that mycotoxins were being used in Afghanistan. We have now concluded that trichothecene mycotoxins have been used by Soviet forces in Afghanistan since at least 1980.

A number of reports indicate that chemical attacks are continuing in 1982. While we cannot substantiate every detail, the pieces of evidence in these reports add up to a consistent picture. For example, a physician in a facility treating casualties among the *mujahidin* (resistance fighters) has reported that he treated 15 *mujahidin* for red skin lesions that he said were caused by Soviet chemical attacks in Qandahar Province in May-June 1982.

TABLE 1

Afghanistan: Reported Attacks, 1982

Date	Village Location	Method of Attack	Form of Materiel	Persons Killed	Persons Taken Ill
early Feb.	north of Shindand	aircraft	yellow substance	4	?
Feb. 4, 5	south of Shindand	helicopter	yellow substance	0	?
Feb. 19	Badakhshan Province	aircraft	yellow crystals	?	?
May-June	Qandahar Province	helicopter rockets	black, yellow, white gases	3	15
June	Farah Province	aircraft bombs	red, black, white smoke	?	?
June 11	Qandahar Province	aircraft bombs	?	15	30
June	Baghlan Province	helicopter	?	?	?
July	Panjsher Valley	aircraft	nerve gas	0	0
Sept. 13	Lowgar Province	pumped from armored vehicle	gas	73	0
Sept. 20	Lowgar Province	pumped from armored vehicle	gas	?	?
Sept.	Lowgar Province	?	?	7	?
late Sept./ early Oct.	Baghlan Province	aircraft bombs	?	?	?

? = Undetermined

Three *mujahidin* died within 12 hours of one attack in the general area of Maharijat south of Qandahar. The *mujahidin* claimed that Soviet helicopters fired rockets that emitted black, yellow, and white gases on impact. The physician said that the surviving victims failed to respond to conventional medical treatment.

We have received reports that on September 20, 1982, Soviet soldiers poisoned underground waterways in Lowgar Province south of Kabul where the *mujahidin* were hiding. According to a *mujahidin* commander in Pakistan, a similar event occurred in the same province on September 13, 1982, resulting in the deaths of 60 adults and 13 children. These two independent accounts described a Soviet armored vehicle pumping a yellow gas through a hose into the waterways.[1]

[1]We know from other sources that Soviet chemical agent delivery methods include this technique, as reported, for example, by a Cuban emigre trained by the Soviets in the use of chemical weapons.

According to the accounts of the September 1982 attacks, the victims' bodies decomposed rapidly, and the flesh peeled away when attempts were made to move the bodies. Since 1979, *mujahidin* resistance leaders, refugees, journalists, and Afghan defectors have described chemical attacks causing almost identical symptoms. Most reports have described the skin as being blue-black after death. Although such symptoms seem bizarre, the large number of reports from a variety of sources since 1979 suggests that they cannot be dismissed (see our March 22 report, p. 16).

In 1982, a Soviet soldier who defected to the *mujahidin* said in an interview with a British journalist that a Soviet toxic agent, termed "100 percent lethal," causes the flesh to become very soft. The Soviet defector stated that the Soviets maintained stores of "picric acid" (probably chloropicrin, a potentially lethal tear gas), the "100 percent lethal" agent, and an incapacitating

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agent near the cities of Qonduz and Kabul. The defector also reported that:

- Unidentified toxic agents had been used in June 1982 on the highway between Termez and the Salang Pass north of Kabul;
- The "100 percent lethal" agent was delivered by rockets; and
- "Picric acid" and an incapacitating agent were delivered by air-dropped canisters.

The defector stated that the Soviets have been preoccupied with protecting the roads and that chemicals were sprayed by planes along the areas adjacent to highways. Chemical grenades reportedly have been used, but the data are inadequate to allow us to hypothesize about the contents, although some symptoms are indicative of mycotoxins.

The reports of rapid skin decomposition as quickly as 1–3 hours after death continue to concern us. There is no recognized class of chemical or biological toxin agents we know of that could affect bodies in such a way. If we assume occasional inaccuracies in reporting by journalists and survivors of attacks, it is possible that phosgene or phosgene oxime could cause such effects after 3–6 hours but with much less softening of tissues than is consistent with stories of "fingers being punched through the skin and limbs falling off." The reported medical effects of other toxic agent attacks are consistent with use of the nerve agent tabun. We have information that both phosgene oxime and tabun are stored by the Soviets in Afghanistan.

The British journalist, who interviewed the Soviet defector, also reported on two attacks described to him by the *mujahidin*, which have not yet been confirmed. One was an attack in the spring of 1982 on Kaiba, where Soviet soldiers reportedly shot victims already rendered unconscious by a gas; the other was in the summer of 1982 near Herat where Soviet troops reportedly loaded the bodies from a gas attack onto a truck and removed them. Reliable information indicates that the Soviets used chemical bombs against *mujahidin* forces in late September 1982 and in early October 1982 in Baghlan Province.

Our earlier findings are reinforced by several reports received this year about earlier attacks not covered in our March report:

- According to a former Afghan Army officer, in September 1981 a Soviet helicopter sprayed a yellow mist in Paktia Province (Sheikn Amir) causing 16 deaths. The survivors had bloody noses and tears; extensive bleeding was reported in those who died. The Afghan officer described a similar attack in Nangarhar Province during the same month in which four persons were killed.
- In early December 1981, according to interviews with survivors, 15 refugees attempting to escape to Pakistan were attacked by a helicopter using gas; four or five people were killed (the youngest and the eldest), while the rest became unconscious for 5–6 hours. The attack occurred about 60 kilometers northwest of Jalalabad.
- According to a Soviet soldier who served in Afghanistan in 1980 and personally observed the use of chemical weapons, the Soviets dispersed chemicals from fighter-bombers and assault helicopters. He said that an aircraft or helicopter first would drop a container and then, on a second run, drop a bomb, resulting in a mixture of two different chemicals that killed everything within the contaminated area. We believe that the soldier may have been describing the delivery of two separate chemical agents, an occurrence described by other eyewitnesses.
- An Afghan veterinarian recently has described an incident in May 1979 in which 20 people and a number of sheep were killed near Qandahar. Soviet lab technicians explained that the incident resulted from anthrax, but the doctor knew that the explanation did not fit the effects observed. Local Afghans told the veterinarian that Soviet vehicles had been in the area spraying a yellow/white powder before the incident.
- In June 1980, an airport official described seeing 200–300 gas containers painted in greens and browns at Qandahar Airport. The containers averaged 35–40 inches high and 26–30 inches in diameter. A subordinate reported three types of gases in the containers: one causing burning in the throat as well as suffocation; one causing what looked like smallpox and blistering; and one making victims tired and sleepy and unable to run or fight. Further, the subordinate stated that the containers were placed in special casings that were dropped from aircraft and exploded on impact, emitting a large cloud of smoke, usually yellow but sometimes other colors. He said he had heard *mujahidin* describe these gas attacks and had himself seen animals that had been killed by the gases. We lend credence to this report because we know from other evidence that chemicals are stored at Qandahar Airport, which is an important staging area for Soviet military operations.
- Finally, information received this year revealed that a Soviet adviser inspecting sites for housing Soviet troops before the Afghan invasion indicated that Soviet chemical defense forces entering Afghanistan would bring in extensive stores of toxic materials. The adviser indicated that a proposed garrison in Kabul would be inappropriate for the Soviet chemical defense unit because the materials it transported could devastate the city if an accident occurred.

Laos

H'Mong refugees, recounting details of toxic agent attacks and exhibiting severe medical symptoms from exposure to the agents, fled to Thailand every month from January through June 1982. They brought out more samples contaminated by a yellow, sticky substance described as a "yellow rain" dropped by aircraft and helicopters on their villages and crops. We have preliminary reports on attacks as recent as October 1982. We now know that the yellow rain contains trichothecene toxins and other substances that cause victims to experience vomiting, bleeding, blistering, severe skin lesions, and other lingering signs and symptoms observed by qualified physicians. Experts agree that these people were exposed to a toxic agent and that no indigenous natural disease, plant, or chemical caused these unique physical effects.[2]

Laboratory analyses of blood samples from these victims and studies on experimental animals have shown that trichothecene toxins are retained in the body for much longer periods of time than previously thought. Scientific research has shown that the multiple-

[2]See Canadian report to the United Nations: "Study of the Possible Use of Chemical Warfare Agents in Southeast Asia," Dr. H. B. Shiefer, University of Saskatchewan.

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phase distribution pattern in animals includes a secondary half-life of up to 30 days. We believe that the severe skin lesions observed on victims by doctors are also relevant. Victims whose blood proved on analysis to have high levels of trichothecene mycotoxins exhibit such skin lesions.

Descriptions of the 1982 attacks have not changed significantly from descriptions of earlier attacks. Usually the H'Mong state that aircraft or helicopters spray a yellow rain-like material on their villages and crops. In some reports the symptoms are similar to those described in our March 22 study, and we attribute them to the use of trichothecene toxins. However, in many cases there was no bleeding, only abdominal pains and prolonged illness. These symptoms, described in previous years, suggest that another agent or combination of agents is still being used. The explanation is complicated because different symptoms are ascribed to men, women, children, and animals. It is possible that different agents, lower concentrations of the same agents, or climatic conditions have affected the efficacy of the agents.

Medical personnel in Lao refugee camps in Thailand were much better organized in 1982 to screen victims than in past years. Doctors now routinely use extensive questionnaires and conduct comprehensive medical examinations, including some onsite, preliminary blood analysis. Skilled paramedical personnel oversee preparation of blood and serum samples for proper transport and shipment to the United States or other countries for chemical analysis. Some patients with active symptoms are now being monitored extensively over time.

A number of blood samples have been collected from Laos for analysis in the United States. All biological specimens were drawn by qualified medical personnel, and samples were refrigerated until analyzed in the United States. Analysis of these samples shows that trichothecene mycotoxins continue to be used against H'Mong villages. In addition to blood and urine specimens from victims exposed to chemical warfare, we have collected additional physical samples this year. These physical samples consist of more residue of yellow rain containing mycotoxins from the same attacks that yielded human biological specimens positive for these same toxins. (See Annex A.)

TABLE 2

Laos: Reported Attacks, 1982

Date	Village Location	Method of Attack	Form of Material	Persons Killed	Persons Taken Ill
Jan. 3, 6, 11	Phou Bia*	helicopter spray	yellow rain	0	?
Jan. 4	Phou Bia	aircraft	green chemical**	?	?
Jan. 9	Phou Bia	artillery	white/yellow cloud	0	?
Feb. 13	Phou Bia	aircraft spray	yellow rain	0	?
Feb. 21, 22	Phou Bia	helicopter	white powder	0	?
Feb. 28	Phou Bia	helicopter	white powder	10	30
Mar. 10	Phou Bia	helicopter, aircraft	red, yellow/ white clouds	many	many
Mar. 17	Phou Bia	helicopter spray	yellow rain**	20	?
Mar. 25	Phou Bia	helicopter spray	yellow rain**	1	40 families
late Mar., early Apr.	Phou Bia	aircraft	yellow rain**	27	many
Apr. 1	Phou Bia (3 villages)	?	yellow rain	many	4
Apr. 17, 18, 30	Phou Bia	aircraft	yellow rain	10	?
Apr./May	Phou Bia	aircraft, helicopters	yellow rain	0	many
May 20	Phou Bia	aircraft	yellow rain	4	100
May 24	Phou Bia	aircraft	yellow rain	9	many
May	Phou Bia	polsoned river	?	0	many
June 17	Phou Bia	aircraft spray	yellow rain	4	many

? = Undetermined

*Phou Bia refers to mountain area where H'Mong villages are located.

**Samples from this attack contained mycotoxin (see Annex A).

The number of reported attacks in Laos in 1982 did not differ significantly from the frequencies reported for comparable periods in the years 1977 through 1981. Reported fatalities per attack during 1981 and 1982 showed an apparent decrease, suggesting the possibility that less lethal toxic agents, or lower concentrations of the same agents, are now being used. This apparent decrease, however, was not statistically significant and could have been caused by a number of other factors, including the following:

- Due to emigration and the high number of fatalities since at least 1976, the H'Mong were living in smaller, more scattered communities.
- H'Mong survivors still in Laos were warier and quicker to take cover and to use rudimentary protective measures at the first sign of attack.
- The H'Mong were not taking time to count victims—this is supported by the existence of very few reports that indicate the precise number of people affected by a toxic agent attack.

As stated in the March report, the Soviet Union maintains in Laos significant numbers of advisers who provide maintenance assistance, technical support, and training in both conventional and chemical warfare. A former Lao transport pilot who defected early this summer has described the aerial movement, under Soviet supervision, of toxic agents within Laos.

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Kampuchea

Most reports of toxic attacks in Kampuchea for the period 1978–June 1982 come from Democratic Kampuchean (DK) sources, including interviews with DK military personnel. Evidence from other sources confirmed most of these reports. In 1982, most reported attacks occurred near the Thai border, making it easier to obtain samples and other direct evidence of toxic agent use.

In the first 6 months of 1982, the number of reported toxic agent attacks in Kampuchea was about half the number reported during the same periods in 1980 and 1981. The number of reported deaths per attack also decreased, but data were insufficient to determine if this decrease was statistically significant. We also have preliminary reports on attacks through early November 1982.

In February and March 1982, several attacks occurred just across the Kampuchean border in Thailand. Analysis of samples collected from the attacks was performed in Canada, Thailand, and the United States. Although differing sampling techniques give rise to significant sampling error and lead to slightly different analytical results, both the U.S. and Thai analysts, using different analytical techniques, found trichothecene mycotoxins in their samples.[3] The Canadian team investigating these attacks has published a detailed medical assessment of the victims' symptoms; it concluded that illness had in fact occurred and was caused by a toxic agent, although preliminary tests for trichothecenes proved inconclusive in the Canadian sample.

Blood and urine samples from Kampuchean victims of a toxic agent artillery attack on February 13, 1982, contained trichothecene toxins (Annex A). In addition, post-mortem tissue from a victim of this same attack confirmed the presence of trichothecene toxins (Annex B). Analysis of additional samples showing the presence of trichothecenes taken from other attacks is also found in Annex A.

The Vietnamese conducted toxic agent attacks this year against another resistance group, the Kampuchean People's National Liberation Forces. On several occasions in March-May 1982, the resistance camp at Sokh Sann was hit with toxic artillery shells and bombs. Samples of contaminated vegetation and yellow residue from these attacks

TABLE 3

Kampuchea and Thailand: Reported Attacks, 1982

Date	Village Location	Method of Attack	Form of Material	Persons Killed	Persons Taken Ill
Kampuchea					
Feb. 13	border near Khao Din	artillery	?*	1**	100
Feb. 23	border near Pailin	spread along border	yellow powder	0	?
Mar. 3	Battambang Province	artillery	?	0	?
Mar. 5, 7	Pailin area	aircraft spray, artillery	white powder*	0	10
Mar. 7–11	Sokh Sann	artillery, ground spray	yellow substance	0	many
Mar. 10	Battambang Province	aircraft	toxic substance	25	12
Mar. 10–13	Battambang Province	aircraft, artillery	toxic substance	30	?
Mar. 17	Sokh Sann	artillery	yellow/white powder	0	many
Mar. 24	Battambang Province	poisoned water	yellow powder	4	?
Apr. 29	Battambang Province near border	aircraft spray	yellow powder	3	7
May 23, 26	Sokh Sann	aircraft spray	?	0	?
June	Preah Vihear Province	poisoned food and water	?	2	many
June 24	border near Nong Chan	mortar	yellow cloud	0	4
Thailand					
Feb. 19	Pong Nam Ron District	aircraft spray	yellow powder*	0	many
Mar. 3	southeast of Pong Nam Ron District near border	aircraft (powder wind-blown over border)	powder	0	many
Mar. 5	Pong Nam Ron District	mortars	gray/black smoke	0	18
Mar. 6, 8	southeast of Pong Nam Ron District near border	aircraft spray	yellow powder	0	many

? = Undetermined

*Samples from this attack contained mycotoxin (see Annex A).

**See Annex B for detailed analysis of autopsy results of the victim.

[3]It was thought initially that a harmless yellow powder had been dropped on Thai villages as part of a disinformation campaign attempting to discredit U.S. sample analysis results. Within days of such an attack, the Thai Ministry of Health announced that only ground-up flowers had been found. However, Thai officials later stated that further analysis showed traces of toxin and that the earlier Health Ministry announcement was based on incomplete investigation.

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are now being analyzed. Attacks occurred in Kampuchea through June 1982, providing new samples; qualitative tests indicate that the presence of trichothecenes is probable. The results of confirmatory analyses are pending.

Several Vietnamese military defectors from Kampuchea have provided valuable information in 1981 and 1982 on chemical weapons use and on the Vietnamese chemical warfare program and have reported that some types of agents are supplied by the Soviet Union. Information from other sources also confirms our earlier view that the Vietnamese possess toxic agent munitions and are equipping their own troops with additional protective equipment.

ANNEX A

Analysis of Samples for Chemical Warfare Agents and Toxins

The identity of the agents and toxins being used in Laos, Kampuchea, and Afghanistan cannot be determined without collection and analysis of at least one of the following: environmental or physical samples contaminated with agent, the munitions used to deliver agents, or biological specimens from attack victims.

The likelihood of detection of chemical agents and toxins in contaminated samples depends on a number of factors. These include the persistency of the agent; the ambient temperature; rainfall; wind conditions; the media on which the toxic agent was deposited; and the time, care, and packaging of the sample from collection to laboratory analysis. Many standard chemical warfare agents and toxins disappear from the environment within a few minutes to several hours after being dispersed. These include, for example, the nerve agents sarin and tabun, the blood agents hydrogen cyanide and cyanogen chloride, the choking agents phosgene and diphosgene, and the urticant phosgene oxime. Other standard agents—such as the nerve agents VX and thickened soman and the blistering agents sulfur mustard, nitrogen mustard, and lewisite—may persist for several days to weeks depending on weather conditions. The trichothecene toxins are stable under laboratory conditions but degrade in field samples due to metabolism by micro-organisms contained in the sample. To maximize the chances of detection, sample collections should be made as rapidly as possible after a toxic agent assault; with many agents this means minutes to hours. Given the situation in Southeast Asia and Afghanistan, this usually has not been possible. Although numerous samples were collected, few held any realistic prospect for yielding results. However, when immediately collected and properly handled and using the advanced technology now available, trichothecenes may be detected in both physical and biological materials up to several months after the attack.

Samples have been collected from Southeast Asia since mid-1979 and from Afghanistan since May 1980. To date, more than 350 individual samples—of greatly varying types and utility for analytical purposes—have been collected and analyzed for the presence of traditional chemical agents. About 100 additional samples are pending completion of analysis. All environmental and nonmedical samples were submitted to the U.S. Army Chemical Systems Laboratory for analysis for traditionally recognized chemical warfare agents and other toxic materials. Tissue specimens and body fluids from attack victims were submitted to the Armed Forces Medical Intelligence Center. Under the sponsorship of that organization, the biomedical specimens were analyzed for the presence of trichothecene mycotoxins and other toxins by Dr. Chester Mirocha, University of Minnesota; Dr. Joseph Rosen, Rutgers University; and Dr. Tim Phillips, Texas A&M University.

To date, biomedical samples (blood, urine, and/or tissue) from 33 alleged victims have been screened. Specimens from 16 of these individuals show the presence of trichothecene mycotoxins. In addition, six environmental samples from alleged attack sites have been analyzed by Dr. Mirocha. Five show the presence of unusually high concentrations and combinations of trichothecene mycotoxins.

Evidence of Chemical and Toxin Agent Use in Southeast Asia and Afghanistan, 1982

Sample	Result
Laos	
blood	trichothecene toxin
blood (post-mortem)	trichothecene toxin
yellow residue	trichothecene toxin
vegetation	trichothecene/ aflatoxin B1
Kampuchea/Thailand	
blood	trichothecene toxin
urine	trichothecene toxin
tissue (autopsy)	trichothecene/ aflatoxin B1
yellow residue	trichothecene toxin
vegetation	trichothecene toxin
water	trichothecene toxin
water	cyanide
Afghanistan	
gas mask (two sets)	trichothecene toxin
vegetation	trichothecene toxin

Details concerning samples analyzed since the March report—including the circumstances of their collection, analysis, and the results—are provided in this annex. Results of analysis of earlier samples were included in our March 22, 1982 report.

POSITIVE SAMPLE RESULTS

Afghanistan

One-quarter of the external surface of a Soviet gas mask, obtained near Kabul in September 1981, was recently processed for analysis, employing techniques not previously used, and showed the presence of T-2 toxin. This analysis has been verified by two other laboratories. Similar analysis of material from the hose connections of another Soviet gas mask removed from a dead Soviet soldier in December 1981 in Afghanistan is indicative of the presence of the trichothecene toxins, T-2, DAS, verricarol, and another type of mycotoxin—zearalenone. It is believed that these

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protective masks were worn during operations in which a toxin agent was used.

Preliminary analysis of a third sample acquired in February 1982 also indicates the presence of trichothecenes.

Laos

1) Blood samples were drawn from an ill H'Mong couple on March 21, 1982, by a physician in the Ban Vinai refugee camp. The victims were exposed to toxin agent attacks on November 11, 1981, and January 4, 1982. They remained ill and under treatment on March 21, 1982, when blood samples were obtained. During the November 1981 attack, an aircraft sprayed a yellowish agent. Although no one died in the village, symptoms such as bloody diarrhea were experienced by most of those exposed. In the January 1982 attack, a greenish chemical was sprayed from an airplane. Vomiting with blood, bloody diarrhea, blurred vision, chest pain, eye irritation, and skin rash were reported. Lingering effects included rash, pain in the joints, and fatigue. The blood samples were analyzed for three trichothecene toxins: DAS, T-2, and HT-2. The blood sample from the male was found to contain 13.5 ppb T-2 toxin. The female was negative for all toxins analyzed.

2) Three blood samples were drawn by a U.S. physician on April 17, 1982, from three H'Mong refugees: Bloc Her, an 8-year-old boy; Tong Her, a 6-year-old boy; and Xia Sue Xiong, a young girl. They were among a group exposed to a toxic agent attack in late March 1982 in Laos. The agent used was described as being yellow to reddish brown. It was sticky and dried to a powder. Bloc Her had been severely ill with bloody diarrhea and coughing of blood. Xia Sue Xiong was suffering from bloody diarrhea and abdominal pain. The blood samples were analyzed for three trichothecene toxins: DAS, T-2, and HT-2.

Trichothecene Toxin Analysis

Victim	DAS	T-2	HT-2
Bloc Her	negative	negative	negative
Tong Her	negative	110 ppb	296 ppb
Xia Sue Xiong	negative	46 ppb	negative

ppb = parts per billion

3) Post-mortem blood samples were taken from a 25-year-old H'Mong refugee who had been admitted earlier to a refugee hospital at Ban Vinai, Thailand. Just before death he had suffered from a massive gastrointestinal hemorrhage. He had claimed exposure to a toxic agent attack sometime earlier in Laos. The blood was drawn in the hospital on April 17, 1982, and analyzed for three trichothecene toxins: DAS, T-2, and HT-2.

Trichothecene Toxin Analysis

Specimen Type	DAS	T-2	HT-2
heparinized blood	negative	15 ppb	19 ppb

4) Blood was drawn on April 6, 1982, from Neng Xiong, a H'Mong refugee in Thailand. She was suffering from the effects from a toxic agent attack that occurred in Phou Bia, Laos, on March 25, 1982. The entire population of the village (40 families) suffered from vomiting, fever, backaches, headaches, and chest pain after a helicopter dropped a yellowish agent. Many villagers also developed swollen eyes. It was reported that one woman and several animals died. Neng Xiong's blood was analyzed for three trichothecene toxins: DAS, T-2, and HT-2.

Trichothecene Toxin Analysis

Specimen Types	DAS	T-2	HT-2
heparinized blood	negative	100 ppb	8 ppb
non-heparinized blood	negative	33 ppb	34 ppb

5) A H'Mong refugee reported being subjected to a toxic agent attack on March 17, 1982, in Phou Bia, Laos. The agent, which "looked like yellow rain," was sprayed by a helicopter at low altitude. The sticky yellow spots dried to a powder in 3-4 hours. Immediately after the attack, the victim developed stomach and chest pains and vomited. Vomiting with blood began later and continued over the next 24 hours. Other symptoms included headache, shortness of breath, dizziness, eye irritation, and vision disturbances. The refugee also developed a rash and blisters. Blood samples were drawn by a physician at Ban Vinai refugee hospital in Thailand on March 31, 1982. The samples were analyzed for three trichothecene toxins: DAS, T-2, and HT-2.

Trichothecene Toxin Analysis

Specimen Types	DAS	T-2	HT-2
heparinized blood	negative	19 ppb	negative
non-heparinized blood	negative	3 ppb	2 ppb

6) Results of analysis of two environmental samples from attack sites in Laos were reported in our March 22 report and are not repeated here. An additional set of environmental samples taken from an allegedly contaminated area in Laos near Phu He was obtained for analysis. Although symptoms were manifested in individuals collecting and handling the sample, no trichothecenes were detected upon analysis.

Kampuchea/Thailand

1) On February 13, 1982, at least 100 Kampuchean soldiers were subjected to an artillery-delivered toxic attack by Vietnamese forces and became ill. The attack took place near the village of Tuol Chrey in an area north of Khao

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Din, about 300 meters from the Thai-Kampuchean border. One individual subsequently died (see autopsy results, Annex B). Reported symptoms included burning eyes, blurred vision, shortness of breath, chest pains, vomiting, and vertigo. Some victims also trembled and generally felt weak. Blood and urine samples were taken from a number of victims at various times after the attack as well as from a control group of individuals living under similar conditions but not subject to the toxic agent attack. Blood and urine samples from the control group were negative for all analyzed toxic agents, including trichothecene toxins.

On February 14, 1982, 1 day after the attack, blood samples were taken from two victims: Pen Nom and Prek Reth. On February 15, a urine sample was taken from Pen Nom, while on February 16, a urine sample was taken from Prek Reth. Both blood and urine samples were analyzed for the presence of the trichothecene mycotoxins T-2 and HT-2.

Trichothecene Toxin Analysis

Victim	Specimen	T-2	HT-2
Prek Reth	blood	18 ppb	22 ppb
	urine	negative	negative
Pen Nom	blood	11 ppb	10 ppb
	urine	trace	18 ppb

On March 4, 1982, 19 days after the incident, some victims still showed effects of the attack and were being treated in Nong Pru hospital in Kampuchea. Further blood samples were drawn at that time from Prek Reth and five additional victims.

Trichothecene Toxin Analysis

Victim	T-2 Toxin
Prek Reth	negative
Kim Ving	7 ppb
Mau Sereth	negative
Seng Nem	negative
Ching Soeum	negative
Chem Ron	3 ppb

Analysis of tissue samples from a victim of the February 13 attack is described in Annex B.

2) On March 5, 1982, a small Vietnamese aircraft sprayed a white powder in an area near Pailin, Kampuchea. On March 6, 1982, 10 of a group of 15 Kampucheans were unable to continue walking due to illness after passing through the area. Symptoms included nausea, vomiting, shortness of breath, blurred vision, diarrhea, bloody discharge from the nose, and burning sensation in the chest and abdomen. A second attack occurred on March 7, 1982, when some of the same Kampucheans were subjected to Vietnamese toxic artillery shelling. The agent produced nausea, dry mouth, and blurred vision and also caused loss of consciousness and muscle twitching. Despite medical treatment, a number of the victims died.

Samples were taken from three survivors exposed to the contaminated area on March 6 and 7. Blood and urine were taken on March 13, 1982.

Trichothecene Toxin Analysis

Victim	Specimen*	DAS	T-2	HT-2
Neung Hon	urine	negative	5 ppb	2 ppb
	blood	negative	7 ppb	negative
Chan Saran	urine	negative	4 ppb	1 ppb
	blood	negative	negative	8 ppb
Bun Thoeum	urine	negative	22 ppb	7 ppb
	blood	negative	negative	negative

*Blood samples were heparinized.

3) A sample of contaminated vegetation was obtained following spraying by a Vietnamese aircraft in Pong Nam Ron District near the Thai-Kampuchean border on February 19, 1982. Analysis of this sample for known chemical agents was negative. However, the trichothecene toxin T-2 was present at a level of 86 ppb. DAS was also present at 30 ppb. The sample was of insufficient size to analyze accurately for the toxins nivalenol and deoxynivalenol.

ANNEX B

Autopsy Results of a Chemical Warfare Attack Victim in Kampuchea

Chan Mann was one of several victims of a February 13, 1982, toxic agent attack in the area of Khao Din. The victim, being treated at Nong Pru Hospital, reportedly made a brief recovery on March 12 and 13, followed by a relapse when he became anuric, feverish, restless, and slightly jaundiced. On March 16, he lapsed into a coma and died. A urinary catheter was inserted approximately 4 hours before death, but only minimal blood-tinged urine was obtained. Shortly before death the victim vomited blood. Kampuchean physicians performed a necropsy. Tissue sections of heart, esophagus/stomach, liver, kidney, and lung were taken and fixed in formaldehyde. Tissue samples were given to both U.S. and Canadian officials for analysis.

The samples delivered to the United States were submitted to several U.S. laboratories for gross, microscopic, histopathological, and chemical-toxicological analysis.

Results of Analysis of Tissue Samples for DAS, T-2, and HT-2[1]

Material	Amount (g)	Toxins Detected DAS[2]	T-2	HT-2
Heart	7.9	—	—[3]	1 ppm
Stomach	13.5	—	25 ppb	4 ppm
Liver	9.5	—	—	—
Kidney	10.4	3 ppm[4]	7 ppb	—
Lung	4.5	—	9 ppb	—
Intestine	5.3	—	88 ppb	10 ppb

[1]DAS (Diacetoxyscirpenol), a trichothecene toxin; T-2, a trichothecene toxin; HT-2, a metabolic product of T-2.

[2]DAS was used as internal standard—i.e., DAS was added to each tissue sample as a standard to check accuracy of analysis. Only the kidney had a concentration of DAS greater than the amount added.

[3]Toxins were not detected. Concentration of DAS was no greater than the added internal standard.

[4]Endogenous DAS in sample detected in concentration greater than the standard.

Note: Tissues were analyzed for trichothecene toxins by Dr. C.J. Mirocha, University of Minnesota. A parallel analysis performed by Dr. J. Rosen, Rutgers University, also revealed the presence of high levels of trichothecene toxins.

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A high incidence of natural aflatoxin contamination of food in Southeast Asia has been well documented. Linderfelser and coworkers (1974) have shown that aflatoxin and T-2 toxin in combination have a synergistic effect and, therefore, it was of interest to determine the extent of aflatoxin in tissue of this individual.

Results of Analysis of Samples for Aflatoxin

Material	Weight of Sample (g)	Sample	
		Actual (ng/g)[1]	Adjusted[2] (ng/g)
Stomach	3.04	19.8	22.5
Liver	3.00	20.2	23.2
Kidney	7.50	15.3	17.4
Intestine	3.02	11.2	12.7

[1]Nanograms per gram.

[2]Values adjusted on basis of 88% recovery—that portion of aflatoxin found when a known amount is added to the sample.

Note: Aflatoxin analyses were conducted by Dr. Phillips, Texas A&M University.

Levels of aflatoxin detected in the tissues were so high that it seemed prudent to investigate the possibility that this individual exposure to aflatoxin was not due to a natural contamination but may have been related to the chemical attack. To this end, portions of the sample of yellow rain from Laos previously shown to contain 143 ppm of T-2 toxin and 27 ppm of DAS were submitted to Dr. Mirocha and Dr. Phillips for analysis for aflatoxin B1. Independent thin-layer chromatography and high-performance liquid chromatography analyses were negative for aflatoxin, supporting a hypothesis that this toxin is not always a component of a yellow rain sample and that the victim's exposure to aflatoxin may have been due to contamination of the food source. It does not, however, rule out the possibility that aflatoxin is a component of some yellow rain attacks. Preliminary analysis of some more recent yellow rain samples indicates the presence of aflatoxin not consistent with a natural contamination. In any case, the findings of aflatoxin in these tissues is important since the high incidence of exposure to natural outbreaks of aflatoxin contamination in Southeast Asia may induce a greater susceptibility to trichothecenes in this population.

Portions of each tissue sample were submitted to Dr. Charles Stahl, University of Tennessee Medical School, for histopathological examination. A summary of the pathology found included: hemorrhage into the heart tissue with evidence of cell destruction and inflammation, cirrhosis of the liver, hemorrhage and cellular destruction of kidney tubules, hemorrhage in the bronchi, and congestion and destruction of the lung. The details of these results and similar findings by other pathologists are consistent with results of analysis of animals exposed to trichothecenes.

No single post-mortem finding proves cause and effect of toxin exposure and death, but these data taken together provide objective evidence that:

- Reports from witnesses of yellow rain attacks are valid and that bleeding sometimes occurs in the lung, stomach, intestine, and kidney or bladder.
- Persons already debilitated by disease or exposure to other toxins have a greater risk of death from trichothecene toxicosis.
- Microscopic examination shows that tissue damage occurs in humans after moderate-to-heavy exposure to trichothecenes. The damage is similar to that found in experimental animals.
- Microscopic damage persists for 1 month or longer.
- Trichothecenes are known to cause long-term damage to rapidly dividing tissue. These toxins accumulate and persist at least in the organs that were examined.
- Aflatoxin found in the tissues may be foodborne and is not necessarily a component of the yellow rain substance. However, aflatoxins and trichothecene toxins act synergistically, and they could be components of a toxic crude extract mixture. Emerging data from several sources lend credibility to such a hypothesis; therefore, investigation seems warranted.

ANNEX C

Discussion of Analytical Findings[4]

The finding of T-2 toxin and HT-2 toxin in blood, urine, and tissue samples from yellow rain victims is highly significant in view of the fact that no trichothecenes could be detected in similar samples from the control population who were not exposed to yellow rain. The finding of such high levels of trichothecenes weeks after exposure is surprising and raises questions concerning the distribution, metabolism, and excretion of these toxins as well as their long-term effects.

Only limited research concerned with elimination rates of the trichothecenes has been conducted. Ueno, et al., 1971, reported that orally administered fusarenon-x was rapidly distributed to the tissues and reached peak levels by 3 hours after dosing. The kidney was believed to be the major organ of excretion. Matsumoto, et al., 1978, conducted studies with T-2 toxin which led him to conclude that the liver and biliary system were the major organs of T-2 excretion. Chi, et al., 1978, administered oral doses of T-2 to broiler chickens. Peak tissue levels were reached by 4 hours after dosing, and the liver contained the greatest amount of toxin. By 12 hours after dosing, however, the muscle, skin, and bile contained the highest amounts of detectable toxin. By 48 hours, 82% of the administered dose had been excreted. Robinson, et al., 1979, showed that T-2 toxin was excreted into cow milk at levels up to 160 ppb after daily administration of 0.6 mg/kg doses.

Studies concerned with metabolism of the trichothecenes have also been limited in number. Yoshizawa, et al., 1980, reported that in rat liver *in vitro* studies with the S-9 fraction of rat liver homogenates, HT-2 made up 50% of the metabolic products. Other metabolites included TMR-1 (19%), TMR-2 (2%), and T-2 tetraol (4%). In *in vivo* studies, HT-2 was one of the major products eliminated in the excreta of chickens (Yoshizawa, et al., 1980) as well as urine, feces, milk, and blood of dairy cattle (Yoshizawa, et al., 1981).

The finding of T-2 toxin and HT-2 in the blood and tissue of humans weeks after their exposure to the toxins would seem to indicate that enough toxin remains bound in body tissues to allow detection by sensitive instrumentation. Trichothecenes have been shown to bind to ribosomal proteins (Ueno, 1975) and to react with sulfhydryl containing compounds such as glutathione (Foster, et al., 1975) and with proteins such as albumin (Chu, et al., 1979). It would appear that although most of the toxin would normally be expected to be excreted within 48 hours after exposure, small amounts of the toxin and its metabolites remain bound to body tissues for much longer periods. The

[4]Based on excerpts from a paper by Dr. C.J. Mirocha and Dr. S. Watson, presented at an international symposium on mycotoxins in Vienna on September 1, 1982.

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size of the dose administered and the route of exposure may have a significant effect on the proportion bound, since a sudden, rapidly absorbed, massive dose may overload normal excretion and detoxification mechanisms, resulting in greater tissue binding of the toxin. Similar apparent long-term storage of mycotoxins has been reported previously for aflatoxin B. Although most of the administered dose of aflatoxin is rapidly metabolized, Shank, et al., 1971, demonstrated in studies of monkeys that unmetabolized aflatoxin B could be detected up to 6 days after administration of a sublethal dose.

Additional significant findings lie in the trichothecenes found in the leaf samples (T–2, DON, nivalenol) and yellow powder (T–2, DAS). The concentrations found and their combination are not normally found in nature and it would appear that these mycotoxins found their way into the environment by the intervention of man. The most compelling evidence is the presence of T–2 and DAS in the yellow powder. Both toxins are infrequently found in nature and rarely occur together. In our experience copious producers of T–2 toxin (*F. tricinctum*) do not produce DAS and, conversely, good producers of DAS (*F. roseum* 'Gibbosum') do not produce T–2. This is also supported by our experience that a good producer of DON does not produce T–2 or DAS but could produce nivalenol. Thus, we have more than just the quantity of toxins produced to explain, but also the kinds that respective species and their isolates produce. Theoretically, it is possible to genetically manipulate or select an isolate that would produce copious amounts of two or more of these toxicants, but this would require a sophisticated research effort and sophisticated technology based on experience.

It is difficult to explain the presence of trichothecenes on leaf surfaces. Fusarium is not a leaf pathogen, and so we would not expect it to colonize leaves indiscriminately. Fusarium does colonize the roots and vascular tissue (causes wilt diseases) of some plants, and it would have to produce the toxins *in situ* and translocate them to the leaves. This has never been demonstrated in the pathogenesis of Fusarium-infected plants. If a pathogen like *F. oxysporum* f. *lycopersici,* pathogenic to tomatoes, were to produce trichothecenes and translocate them to the leaves, one would not expect such high concentrations and combinations of toxins. Moreover, we are not certain that pathogenic isolates of the latter produce trichothecenes during pathogenesis. It is a well known plant pathological principle that production of toxins by pathogens in laboratory culture does not signify that these toxins also are produced in the host.

Apart from the controversy of the trichothecenes occurring on the leaves, it is difficult to imagine a reasonable explanation for the appearance of T–2 and DAS in the yellow powder. To those who claim that they dropped onto the soil and rocks from overhanging leaves, this is contrary to any known facts about trichothecene occurrence or distribution. The burden of proof remains with those alleging such an unlikely hypothesis.

The finding of T–2 toxin, diacetoxyscirpenol, deoxynivalenol, zearalenone, and *Fusarium* pigments in leaves, water, yellow powder, and fragments originating at sites of yellow rain attacks in Southeast Asia and their absence in background samples (leaves, corn, rice, water, soil) from areas not exposed to yellow rain strongly implicates their use as warfare agents. Moreover, the finding of T–2 toxin and HT–2 toxin (a metabolite of T–2 toxin in animals) in the blood, urine, and tissue of victims of these attacks provides unequivocal evidence of their use as weapons.

* * * *

Details of the experimental procedures used in these analyses were presented at the Society of Toxicology meeting in Louisville, Kentucky, on August 16, 1982; at an international mycotoxin symposium in Vienna, on September 1, 1982; and at the Association of Analytical Chemistry meeting in Washington, D.C., on October 28, 1982.

Two scientific manuscripts describing those analyses have been submitted for publication in refereed journals and other studies pertaining to nongovernmental analyses are in press. ■

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